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		Official page numbers:
SALMONELLA ORANIENBURG, ONTARIO F-1	177-179	**For reference purposes, citing should refer to the page numbers of the printed copy and not to those of the FAX copy (F-#).**
VIBRIO CHOLERAE NON-O1 ON BLOOD CULTURE, SASKATCHEWAN F-3	180-181	
STRAIN CHARACTERISTICS OF *STREPTOCOCCUS INIAE* ISOLATED FROM TILAPIA SPECIES IN VANCOUVER, BRITISH COLUMBIA. F-4	181-182	
EL NIÑO AND ITS HEALTH IMPACTS F-4	182-184	

SALMONELLA ORANIENBURG, ONTARIO

Introduction

Twenty-two cases of *Salmonella* Oranienburg with onset of illness between 12 May and 30 June 1998 were reported to the Ontario Ministry of Health as part of the routine surveillance of enteric pathogens. This is in contrast to 14 and 10 cases reported in Ontario for all of 1997 and 1996, respectively.

Discussion

A case series and case-control study were conducted to determine the source of this cluster. A case was defined as an individual with diarrhea occurring between 1 May and 14 July 1998 and laboratory-confirmed *Salmonella* Oranienburg isolated from stools, blood, or urine. All of the 22 individuals were contacted. Two were not included in the case series; one had a urine culture and no gastrointestinal illness, and the other was a secondary case. Therefore, the following results are limited to 20 cases. The cases ranged in age from 7 months to 82 years (median: 50 years), 65% were female, and they lived throughout southern Ontario. The onset of illness occurred between 12 May and 30 June 1998, with 35% (7/20) occurring during the week of 15 June. Exposure histories revealed that 85% (17/20) had eaten cantaloupe during the 3 days prior to their illness. One case could not remember if cantaloupe was consumed during the time period in question. One of the cases was 7 months of age, and the only raw foods eaten were cantaloupe and banana. None of the cases had any cantaloupe available for microbiologic testing. The cases had purchased the cantaloupe at a number of retail outlets between 18 May and 28 June 1998.

A matched case-control study was completed to test the hypothesis that cantaloupe consumption was associated with illness. The first 14 cases identified in the cluster and 28 geographically matched controls were included in the study. The only food item significantly associated with illness was consumption of cantaloupe during a 3-day period (odds ratio, undefined; 95% CI = 3.2 to ∞; p = 0.0003). Exposure to cantaloupe was defined in various ways, including cantaloupe consumption during a particular 3-day period and during an entire 2-month period; consumption of cantaloupe always remained significantly associated with illness.

Laboratory investigations on the isolates from the 20 cases by phage typing and pulsed-field gel electrophoresis (PFGE) revealed that 19 were indistinguishable. The remaining isolate had a different phage pattern and showed a different PFGE pattern, which was classified as possibly related. This case had consumed cantaloupe prior to becoming ill.

During the time period of interest, Ontario cantaloupes were not in season. Cantaloupes were imported into Ontario from numerous sources including the United States, Mexico, and Central America. An attempted traceback of cantaloupes supplied to the retail outlets identified by the cases could not identify a common supplier.

This is the first time in Ontario that cantaloupe consumption has been associated with *Salmonella* Oranienburg. Poultry and other meat products, eggs, and dairy products are the most commonly implicated foods in *Salmonella* outbreaks[1]. Two previous outbreaks of salmonellosis in the United States have been associated with cantaloupe consumption[2,3]. Outbreaks have also been associated with eating watermelon[4,5]. To decrease the risk of *Salmonella* infection from melons, the Food and Drug Administration in the United States recommends that both retailers

and consumers thoroughly clean melons with potable water before cutting, prepare cut melons using clean and sanitized utensils and surfaces, hold cut melons at ≤ 45° F (≤ 7° C) until served or sold, and limit the display of cut cantaloupes to 4 hours if not kept refrigerated[2].

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Source: *S Deeks, MD, Field Epidemiology Training Program, A Ellis, DVM, Bureau of Infectious Diseases, LCDC, Ottawa; B Ciebin, MSc, Central Public Health Laboratory, Ontario Ministry of Health, Toronto; Canadian Food Inspection Agency, Toronto and Ottawa; R Khakhria, BSc, Dip Bact, National Laboratory for Enteric Pathogens, Ottawa; M Naus, MD, Public Health Branch, Ontario Ministry of Health, Toronto; J Hockin, MD, Director, Field Epidemiology Training Program, LCDC, Ottawa ON.*

Editorial Comment

When this outbreak was recognized in Ontario, no other province or territory reported increased activity of this particular serotype. Of interest, two unrelated outbreaks of infection due to *Salmonella* Oranienburg were identified in other jurisdictions internationally during similar time periods. An outbreak in South Australia was associated with eating a locally manufactured gelato (A. Milazzo, Communicable Disease Control Branch, Department of Human Services, South Australia: personal communication, 1998). An outbreak in Washington State was associated with consuming fresh imported mangos purchased from a particular grocery store chain (M. Goldoft, Washington State Department of Health: personal communication, 1998). In Canada, *Salmonella* Oranienburg is not commonly identified and does not rank amongst those 10 serotypes most frequently isolated. Approximately 0.5% of the 6,587 *Salmonella* infections reported to the National Laboratory for Enteric Pathogens were due to this serotype in 1996. In comparison, in the United States, *Salmonella* Oranienburg was the seventh most commonly identified serotype representing almost 2% of all reported *Salmonella* infections[1]. Despite increasing recognition and publicity about food-borne outbreaks, the numbers of cases of salmonellosis reported per year in Canada generally decreased between 1987 and 1992, and then have remained fairly stable for the past 5 years.

This outbreak reported from Ontario, similar to many other outbreaks of salmonellosis in which fresh produce has been implicated, was identified and subsequently investigated because of the temporal clustering of a rare serotype. The lessons learned from these outbreak investigations together with the recognized changes in food production, and distribution and consumption patterns suggest that fresh produce has enormous potential to cause food-borne disease[2]. For the more common *Salmonella* serotypes, it is much more difficult to distinguish an outbreak from the background of presumably sporadic cases for which no epidemiologic investigations are routinely completed. Therefore, we do not know what proportion of the vast burden of salmonellosis may be attributed to consumption of fresh produce.

From the farm to table, there are multiple opportunities for fresh produce to become contaminated. *Salmonella*, as well as *Escherichia coli* O157:H7, *Campylobacter jejuni*, *Vibrio cholerae*, parasites, and viruses may contaminate fresh produce through the use of raw manure or unpotable water, or contact with animals or potentially tainted surfaces including human hands. In addition, pathogens such as *Listeria monocytogenes*, *Bacillus cereus*, and *Clostridium botulinum* are naturally present in the soil[3]. As cantaloupes are grown on the ground, they are particularly vulnerable to their exterior surface being contaminated by dirt and animal excreta. Following the outbreak of *Salmonella* Chester in the United States which was sourced to cantaloupes from Mexico, the Food and Drug Administration (FDA) isolated numerous serotypes of *Salmonella* from approximately 1% of the rinds of imported cantaloupes and watermelons sampled at the Mexico-United States border[2]. Although much smaller than previous outbreaks traced to cantaloupes, the Ontario outbreak suggests either that the "Melon Safety Plan" which focused on chlorination of water used to wash melons and to make ice for shipping them[4] has not been universally implemented or that it is not totally effective. Certainly once the flesh of the cantaloupe is contaminated with *Salmonella*, sanitization is not effective presumably because of the neutralizing capacity of the juice[3]. Therefore, the FDA recommendations provided above, although not evaluated, are prudent preventive measures in the control of the food handler.

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Case Report

VIBRIO CHOLERAE NON-O1 ON BLOOD CULTURE, SASKATCHEWAN

Introduction

Vibrio cholerae can be divided into three main sub-types: O1 toxigenic, O1 non-toxigenic, and non-O1 strains[1]. The O1 toxigenic strains, such as the El Tor biotype, have been historically associated with large outbreaks of classic cholera symptoms internationally. They produce an enterotoxin responsible for profuse watery diarrhea[1,2]. The O1 non-toxigenic strains have been associated with shellfish along the Gulf Coast of the United States[1]. The non-O1 strains generally do not produce the enterotoxin (although some strains do) and are generally only associated with sporadic and rare outbreaks of diarrheal illness[2]. These organisms are increasingly isolated in Canada from residents with no history of travel outside the country[1]. *V. cholerae* non-O1 are found in water sources worldwide[2,3]. In North America, cases have been primarily associated with consuming raw oysters[3]. Non-O1 may be enteroinvasive (O1 toxigenic *V. cholerae* generally is not) and may be associated with sepsis[3]. Non-O1 has been also been associated with skin and ear infections, and has been isolated from the hepatobiliary system[3]. *V. cholerae* has been shown to survive on the external surfaces of houseflies for up to 7 days[4]. In Canada, from 1 April 1996 to 31 March 1997, isolates from six cases were sent for identification and serotyping to the National Laboratory for Enteric Pathogens; all isolates were identified as *V. chlorae* non-O1. An additional six isolates were identified as *V. chlorae* non-O1 during the 1997-1998 period. The majority of these non-O1 cases originated from within the country (D. Woodward, National Laboratory for Enteric Pathogens, Winnipeg: personal communication, 1998).

Case report

On 7 August 1998, a 75-year-old Saskatchewan woman visited the Moose Jaw Union Hospital complaining of an insect bite to her lower left leg earlier that day. Prior to being bitten, she had been wading in a lake in the district. On examination, she was febrile and had findings of cellulitis on her left leg. The patient had a prior history of a wound infection in the same leg 2 years earlier, caused by an abrasion from a tree branch. She also had a history of macular degeneration, osteoarthritis, hypothyroidism, and a remote history of melanoma. Blood cultures were taken at the time and she was placed on empiric therapy of oral Cipro®. She returned to emergency on 9 August complaining of worsening symptoms. The leg was increasing in redness and swelling, but she was afebrile. She was switched to a daily regime of intravenous Ancef® and Rocephin®, and oral Flagyl®, to be given at home. On 14 August 1998, she returned to emergency again, with nausea and vomiting; this was assessed as being related to the antibiotics. She was given Gravol® and sent home.

Public-health response

On 14 August 1998, Moose Jaw-Thunder Creek Health District was notified with the preliminary result of *V. cholerae* from her blood culture. The sample was forwarded to the Laboratory Centre for Disease Control for typing and confirmation and was found to be *V. cholerae* non-O1. While awaiting the confirmation, the patient was contacted and recommendations about hand washing were reinforced. The family physician elected to put the patient on doxycycline for cholera prophylaxis. Stool samples were obtained from her and her husband; they were negative for *V. cholerae.*

The risk-factor history revealed travel to Arizona in the winter of 1997-1998; prior lifetime travel to France, the United Kingdom, Germany, and Sweden (no travel anywhere else); drinking water from a chlorinated municipal source; wading in lake water as described above; no consumption of shellfish (because of allergy); consumption of fish (described as "very well-cooked") in Arizona. Other possible risk factors included an exposure to Japanese tourists who stayed with her family from 4-7 August 1998. The tourists were not ill at the time. An exchange student from Colombia was staying with her daughter for the month prior to her illness, and the patient had eaten at the home once during the 3 weeks prior to her illness. Also, her grandchildren had visited her at her home during the 2 weeks prior to her illness. These grandchildren had been in the Solomon islands a year previously, and during the trip one had been ill with severe diarrhea. Stool samples were obtained from the grandchildren and the exchange student; they were negative for *V. cholerae.*

Discussion

Given that *V. cholerae* non-O1 has been identified as able to exist in the waters of the world, the most plausible explanation of this case is that it was the result of a cellulitis obtained through contact with lake water; the insect bite occurred after she was in the water and may have introduced the organism through the protective lining of the skin. In Saskatchewan in the last 10 years, at least one case of *V. cholerae* non-O1 has been diagnosed a year; all of these cases have had an exposure to lake water. Most isolates were from stool samples associated with diarrheal illness, but one case was an ear infection (Dr. E. Chan, Saskatchewan Health Provincial Laboratory, Regina: personal communication, 1998). The absence of any convincing risk factors of any other nature points to this cause. The potential for severe complications from such infections in the elderly and immunocompromised exists. Closer surveillance of the situation here in Saskatchewan may identify the extent of risk to this particular population.

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Source: *R Ramsingh, MD, Medical Health Officer, Moose Jaw-Thunder Creek Health District, Moose Jaw SK.*

STRAIN CHARACTERISTICS OF *STREPTOCOCCUS INIAE* ISOLATED FROM TILAPIA SPECIES IN VANCOUVER, BRITISH COLUMBIA

Following the 1995-1996 Toronto epidemic of human infection with *Streptococcus iniae* in patients handling fresh whole fish[1,2], an epidemiologic and microbiologic study was initiated to determine the possibility of *S. iniae* carriage on Vancouver's fresh fish. Eleven tilapia fish and six other species sharing circulated tank water and purchased live from five fish markets in the Greater Vancouver Regional District, British Columbia, were swabbed and cultured for *S. iniae* on sheep blood agar and colistin-nalidixic acid medium. Seven out of 11 tilapia and four out of six other fish (rock cod, trout, sculpin) were culture positive by standard microbiologic methods. Pulsed-field gel electrophoresis (PFGE) using restriction endonucleases *Sma*I and *Apa*I revealed that Vancouver's *S. iniae* strain was identical to the epidemic strain of human isolates and some, but not all, of the fish isolates from Toronto. In 1997, a Vancouver surveillance identified two cases of human cellulitis and bacteremia caused by *S. iniae* in patients who sustained inoculation injuries while preparing tilapia. In both cases, the *S. iniae* strain was identical to the Toronto outbreak strain identified by PFGE. Whereas fish farms supplying Toronto markets were located in Tennessee, Arkansas, North Dakota, Delaware, and Illinois, the Vancouver tilapia were traced to a fish farm in North Dakota which does not supply Toronto. Our results suggest that a single clone of *S. iniae* has caused disease in geographically remote regions which may or may not be related to a common source, and that this strain may contain a virulence factor which is important for pathogenicity in humans and fish.

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Source: *N Press, MD, E Bryce, MD, G Stiver, MD, Division of Infectious Diseases and Department of Medical Microbiology, University of British Columbia, Vancouver BC.*

International Notes

EL NIÑO AND ITS HEALTH IMPACTS

Over recent years there has been growing interest in links between El Niño (and other extreme weather events) and human health. A number of studies have demonstrated that pronounced changes in the incidence of diseases can occur in parallel with the extreme weather conditions associated with the El Niño cycle.

Infectious diseases

Strong evidence exists of linkages between these weather variations and increases in the incidence of infectious diseases, such as insect vertor-borne diseases (e.g. malaria, Rift Valley fever [RVF]) and epidemic diarrheal diseases (e.g. cholera and shigellosis).

Climatic factors, such as changes in temperature and humidity, are known to be capable of facilitating or interrupting the capacity of insect vertors to transmit disease to humans. Malaria and RVF are two diseases for which substantial documentation in this area exists. Less well documented, but of increasing interest, are the effects of El Niño/ Southern Oscillation (ENSO) climatic phenomenon on dengue. This largely urban disease, present in tropical regions around the world, is spread by mosquitoes that breed in artificial containers. Thus, in addition to climatic factors, changes in domestic water storage practices brought about by disruption of regular supplies, will also influence patterns of transmission.

Malaria

El Niño events have an impact on malaria control in many parts of the world because the associated weather disturbances influence vector breeding sites, and hence the transmission potential of the disease. It has been recognized that many areas experience a dramatic increase in the incidence of malaria during extreme weather events correlated to El Niño. Moreover, outbreaks may not only be larger, but more severe, as populations affected may not have high levels of immunity. Quantitative leaps in malaria incidence coincident with ENSO events have been recorded around the world; such epidemics have been documented in Bolivia, Columbia, Ecuador, Peru and Venezuela in South America; in Rwanda in Africa; and in Pakistan and Sri Lanka in Asia. Historically, in the Punjab region of north-eastern Pakistan, the risk of malaria epidemics increases five-fold during the year following a major El Niño, and in Sri Lanka, the risk of a malaria epidemic increases four-fold during an El Niño year. These increased risks are associated with above-average levels of precipitation in the Punjab and below-average levels of precipitation in Sri Lanka. In South America and Rwanda, heavy rainfall has contributed to major epidemics of malaria. To be able to forecast the impact of El Niño in different endemic areas, control programs need to develop a thorough understanding of how local vector species respond to climate variability, and how a population's immunity and nutritional status fluctuate over time.

To organize a timely and effective epidemic response, malaria control programs need to incorporate surveillance and epidemic control in their everyday activities.

Rift Valley fever

Outbreaks of RVF, a vector-borne disease that principally infects livestock, have occurred in eastern Africa on almost every occasion that there has been excessive rainfall. As a result of the 1997 El Niño, areas of north-eastern Kenya and southern Somalia experienced rainfall which was 60 to 100 times heavier than normal – the heaviest recorded rainfall since 1961. The rains, which began in October 1997 and continued through January 1998, caused RVF virus-infected eggs of floodwater *Aedes* mosquitoes to hatch. In the outbreak of RVF that followed, livestock losses were considerable in the affected regions. Moreover, the estimated toll of human death due to RVF in the region was 200 to 250, while there were an estimated 89,000 human cases of RVF in north-eastern Kenya and southern Somalia. Other areas of Kenya, and the United Republic of Tanzania were also affected with widespread animal infections; however, their impact on the human population was not as great. Preliminary estimates of infections and deaths among animals and humans suggest this may be the largest outbreak of RVF ever reported.

Cholera and other epidemic diarrheal diseases

These are a major cause of morbidity and mortality in many countries. Outbreaks can be related either to floods or drought (floods, for example, contaminate water supply, while droughts make hygiene more difficult and contaminate the water that remains).

There is circumstantial evidence to indicate a close association between weather changes caused by El Niño and cholera. Since September and October 1997, there has been a deteriorating cholera situation in the Horn of Africa. After heavy rainfall and floods, most of the countries in this region reported a dramatic upsurge in the numbers of cases of and deaths due to cholera. In 1997, a total of 40,249 cholera cases with 2,231 deaths were reported in Tanzania alone (compared with 1,464 cases and 35 deaths in 1996). Kenya reported 17,200 cases and 555 deaths and Somalia 6,814 cases and 252 deaths due to cholera in 1997. With the floods continuing in this region and adding to already limited sanitation, poor hygiene and unsafe water, conditions favour the spread of cholera. At the end of 1997, other countries bordering the Horn of Africa, such as the Democratic Republic of the Congo and Mozambique, were reporting increased numbers of cholera cases and deaths. Reported figures through the first 3 months of 1998 already showed 11,335 cases and 525 deaths in Uganda, and 10,108 cases and 507 deaths in Kenya.

In the Americas, the current cholera epidemic has been raging for 7 years and, associated with a major El Niño, the number of cholera cases started to increase at the end of 1997. In 1998, Peru has been suffering from a major outbreak and has already reported, for the first 3 months of 1998, 16,705 cases and 146 deaths. Other countries which are reporting increasing numbers of cholera cases in 1998 are Bolivia, Honduras, and Nicaragua.

A study examining the relationship between sea surface temper- ature and cholera case data in Bangladesh during 1994 documented a close association between those two variables.

Prediction and prevention

Measures to predict and prevent disease outbreaks related to El Niño are increasing. In south-eastern Africa and the Horn of Africa, the regional WHO Cholera Surveillance Teams, warned by early forecasts of El Niño-related extreme weather events in 1997, were able to help reduce the severity of the cholera outbreak in those regions by means of increased monitoring and heightened preparedness of healthcare institutions in the area.

The Southern Oscillation Index has been used to predict the probability of epidemics of vector-borne diseases (such as Murray Valley Encephalitis in Australia). Satellite remote sensing, used to detect areas of abnormal precipitation via increases in vegetation, highlighted exactly those areas which were hit by the RVF outbreak in east Africa in late 1997 and early 1998. Mathematic modeling techniques to predict the spread of malaria into new areas in relation to climate changes are also being used (e.g. in Kenya).

Source: *WHO Weekly Epidemiological Record, Vol 73, No 20, 1998.*

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